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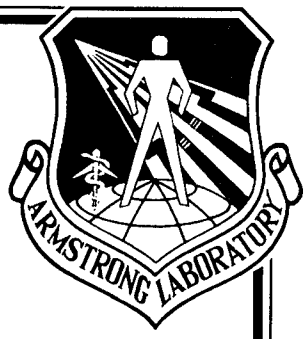

PULSE WAVEFORM AND PULSE AMPLITUDE ANALYSIS DURING LOWER BODY NEGATIVE PRESSURE

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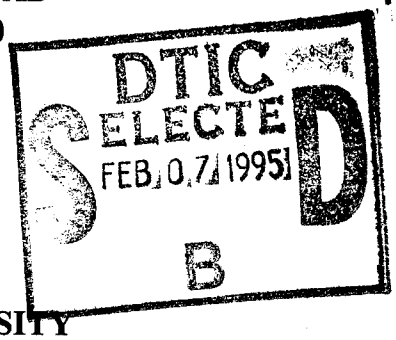

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The voluntary informed consent of the subjects in this research was obtained as required by Air Force Regulation 169-3

This report has been reviewed by the Office of Public Affairs (PA) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

FOR THE DIRECTOR

Thomas J. Moore

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The use of lower body negative pressure (LBNP) as an acceleration pre-conditioning technique for space applications was investigated. The purpose of this study was to evaluate changes in cephalic blood flow during LBNP. The intent was to see if detection or warning of impending syncope was possible, and to simulate effects which occur under exposure to +Gz. Ten subjects underwent the following LBNP profile while in a standing position: Five minutes of baseline at ambient pressure, followed by increments of -10 mm Hg every three minutes to a minimum of -50 mm Hg. They remained at -50 mm Hg for a maximum of twenty minutes or until presyncopal symptoms occurred. An additional five minutes of post-LBNP baseline data were collected. The analog pulse waveform signals were obtained from a pulse oximeter sensor located approximately at eye level on the subject's ear lobe. The pulse waveform analysis included pulse area, amplitude, and duration. Six of the ten subjects experienced presyncopal symptoms during the LBNP profile. Significant changes were observed in several variables at the presyncopal endpoint and included: pulse waveform area ($P=0.0048$), pulse waveform amplitude ($P=0.0236$). In the non-presyncopal group, pulse waveform area ($P=0.0208$), amplitude ($P=0.0070$), and duration ($P=0.0030$) demonstrated significant changes compared to baseline values after exposure to LBNP.

14. SUBJECT TERMS
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PREFACE

Technical support and equipment for this research were provided by the Combined Stress Branch, Armstrong Laboratory, Building 33, Wright-Patterson Air Force Base, Ohio 45433.

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Statistical support and expertise was provided by Chuck Goodyear and Deepa Naishadam of Logicon Technical Services, Inc.

We would also like to thank the members of the Sustained Acceleration Panel for their participation in this study.

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INTRODUCTION

The ability to monitor head-level blood flow may be an important tool in determining a pilot's physiological status in the high acceleration environment. Many studies by Wood et al., have previously demonstrated the use of the "ear opacity" technique during +Gz centrifuge exposure as an effective monitoring modality [14, 15, 16, 17, 18]. The purpose of this investigation was to exploit current off-the-shelf technologies and develop a reliable system to quantify eye level blood flow/pressure.

Previous work conducted at Armstrong Laboratory, Wright-Patterson Air Force Base (WPAFB) in Dayton, Ohio, has demonstrated a change in head-level analog pulse waveform signals obtained from pulse oximeters during exposure of subjects to +Gz on the Dynamic Environment Simulator (DES) centrifuge. Recently, there has been great interest in the performance decrement that occurs prior to full recovery after G-induced loss of consciousness (GLOC) [4]. In addition, there is also great interest in developing methods for detecting impending GLOC and giving feedback to the pilot or crewmember, with the intent of warning or initiating an automatic control system until the pilot recovers [4, 14, 15, 16, 17].

This research study was performed to detect any changes in head-level plethysmographic pulse waveform that occurs during presyncopal lower body negative pressure (LBNP) exposures. LBNP was used with the intent of simulating +Gz [6, 9], and provided the additional benefit of a stationary experimental environment.

Analog arterial pulse waveform data were obtained from a pulse oximeter of a type commonly used to monitor the oxygen saturation and heart rate of clinical patients. In addition, a means of analyzing the pulse waveform in real-time using a microcomputer was developed for this experiment.

BACKGROUND

Lower Body Negative Pressure (LBNP)

Lower Body Negative Pressure (LBNP) is a method in which the abdomen and legs of a subject are exposed to a negative gauge pressure, causing up to one liter of blood to pool in the lower body [10, 11]. The resulting physiological effects are similar to those occurring during hypovolemic shock, high-G acceleration (+Gz), and orthostasis [11, 13].

The application of negative pressure to the body for scientific or medical purposes was first used in 1841 by Junod, who used it to create a localized hyperemia [13]. Junod also suggested that

it could be used prior to invasive surgical procedures, since the syncope it was able to produce was considered a "satisfactory state" prior to invasive procedures [13]. In the early 1950's, there was new interest in this procedure among researchers who used it to investigate the response of peripheral resistance vessels to varying ranges of transmural pressures during orthostasis and acceleration [13]. Major interest in LBNP began in the early 1960's when investigators realized that it caused physiological effects similar to those observed during orthostasis and head-up tilt. Aerospace researchers were also interested in spaceflight-related applications of LBNP because the cardiovascular stresses it imposed were independent of gravity [13]. LBNP also simulates central hypovolemia, allowing the study of acute hemorrhage [13].

Since then, LBNP has been extensively utilized in the aerospace medicine field. Presyncopal LBNP involves exposure of the lower one-half of the body to increasingly negative gauge pressure until the subject experiences symptoms of impending syncope. It is used to study the cardiovascular effects of orthostasis and evaluate subjective tolerance. LBNP is used to simulate +Gz in aerospace medicine research [13]. It has also been studied as a potential countermeasure to negative Gz acceleration [1].

Based on heart rate data, Lategola and Trent estimated that -50 mm Hg supine LBNP was considered to be equal to -40 mm Hg seated LBNP [6]. In terms of blood volume and heart rate displacements, a negative pressure of -50 mm Hg of supine LBNP is considered to be equivalent to +2 Gz [7, 8]. Work by Polese et al., has indicated that +2 Gz and -40 mm Hg seated LBNP resulted in similar changes in heart rate, diastolic blood pressure, and mean arterial pressure [9]. The changes in systolic blood pressure and pulse pressure were more severe with seated LBNP than with +2 Gz [9]. Figure 1 shows a subject wearing the LBNP device.

Pulse Oximetry

Pulse oximeters are commonly used in clinical medicine to non-invasively measure the arterial oxygen saturation and heart rate of patients. Their operation centers on the ability of the hemoglobin molecule to reversibly bind and release oxygen inside erythrocytes. In general, the pulse oximeter sensor consists of an emitter and detector combination. The sensor typically uses two light emitting diodes (LEDs) as light sources and a photodiode as a light detector. One LED transmits red light (wavelength of approximately 660 nm) and the other transmits infrared light (wavelength of approximately 940 nm) [23, 24, 25]. The photodiode is placed on the other side of a pulsating vascular bed (typically across a fingertip or ear lobe) and detects the amount of light that passes through the tissue. Oxyhemoglobin absorbs more infrared light and deoxyhemoglobin absorbs more red

light [24].

Figure 1. LBNP Device

When pulsatile blood flow is not present, the amount of light absorbed will be relatively constant, in the absence of any motion artifact. With each heartbeat, a pulse of blood is propelled past the sensor unit. The inflow of blood will increase the absorption of both infrared and red light, but proportionally more infrared light will be absorbed because the new pulse of blood is particularly high in oxyhemoglobin. The ratio of light absorbed during pulsatile flow to the amount absorbed when pulsatile flow is absent can be calculated. Using a spectrophotometric relationship known as Beer's law, the logarithm of this ratio can be used to determine the oxygen saturation of arterial hemoglobin [23].

In addition to oxygen saturation, pulse oximeters typically display heart rate, which is obtained by analyzing the number of

pulses passing by the sensor over a defined interval of time. Pulse oximeters utilize the principle of plethysmography, defined as "the recording of changes in the size of a part as modified by blood circulation in it," [25]. An analog output of pulse waveform is generated as each pulse of blood absorbs the light emitted by the infrared and visible light diodes. This causes an increased voltage signal output with increased absorption of light and a decreased voltage with decreased absorption, somewhat resembling a sinusoidal curve as each pulse of blood passes by the sensor. The shape and amplitude of this signal will vary directly with the amount of light absorbed, whether it is due to a change in oxygen saturation, a decreased amount of blood, or a change in distance between the emitter and detector. The distance between the sensor's emitter and detector is considered to be essentially constant during use [21]. By using this plethysmographic technique, the oximeter is able to determine the amplitude, configuration, and frequency of a pulse waveform [25].

Because pulse oximeters depend on changes in blood volume to calculate saturation, oximeters with a pulse waveform can also provide a non-invasive means of assessing a patient's intravascular volume status. Blood volume changes with each arterial pulse in the same manner as the intraarterial waveform. Therefore, the pulse waveform can be used to assess changes in pressure and hydration (25).

METHODS

Subjects

Eleven healthy volunteer subjects were recruited from the Sustained Acceleration Stress Panel at Wright-Patterson Air Force Base, Ohio. One subject was unable to complete this study. The ten remaining subjects consisted of 3 females and 7 males. Each subject had been briefed on the medical risks associated with this research and had given informed consent prior to their participation in this study. Table 1 provides an overview of the subject's age.

Table 1. Subject Age Characteristics

	RANGE (years)	MEAN AGE (years)	MEDIAN (years)	S.D. (years)
All Subjects	25-41	30.60	30	5.27
7 males	26-41	32.14	30	5.34
3 females	25-31	27.00	25	3.46

All ten subjects had previous experience with exposure to +Gz on

the Dynamic Environment Simulator (DES), a 19 food radius man-rated centrifuge located at Wright-Patterson Air Force Base. Two of the ten subjects had previously experienced G loss of consciousness while riding the centrifuge.

Materials and Equipment

Two LBNP suits differing in physical size were constructed specifically for this experiment. The two sizes were necessary because of the large variation in physical size of the subjects. The leg sections were constructed out of rigid corrugated PVC tubing which was ten inches in diameter for the small suit and twelve inches in diameter for the large suit. The small suit had flexible knee hinges allowing limited flexion at the knee, which was irrelevant for the purposes of this experiment. The suit design used was similar to a patented version previously used in LBNP experimentation at Wright-Patterson Air Force Base by Tripp et al., [11]. The vacuum source for the LBNP suit was a one-horsepower shop vacuum ("Clements Cadillac Shop-Vac, Model 14, Wet or Dry"). The AC line voltage of this vacuum was controlled using a Variac (rheostat) controller. This allowed fine adjustment of the motor speed, which regulated the amount of vacuum suction and, ultimately, the suit pressure. The LBNP suit was connected to the vacuum source by one inch diameter reinforced tubing. A second port on the suit was used to monitor the negative pressure level.

Continuous monitoring of suit pressure was accomplished with a dedicated pressure transducer, which converted aneroid pressure information to an analog voltage. The output of this device was connected to the microcomputer discussed below and converted to digital form. In addition, the digital pressure output was calibrated to a standard aneroid analog barometer which was connected in-line with the pressure monitoring port at the beginning of each experiment. Both pressure gauges were monitored throughout each experimental run. To ensure accurate pressure readings, the pressure sensing port and vacuum port were on opposite legs of the LBNP suit.

The investigators and subjects each had separate "abort" switches immediately accessible throughout the experimental run. These consisted of a switch on the Variac controller and a switch on a multi-outlet strip.

Continuous ECG recording was accomplished using disposable electrodes in a standard Einthoven Lead II configuration. The ECG monitor used was a Hewlett-Packard model 78304A with memory ("trickle-down") capability. A standard paper strip-chart recorder was attached in-line to allow hard-copy output of ECG signals on demand.

An additional six-channel strip chart recorder was connected in line with the appropriate signals, to allow hard copy output of suit pressure, ECG, and pulse waveform.

A Criticare model 503 pulse oximeter was used in conjunction with a Criticare model 560 interface module to allow output of analog signals. The pulse oximeter sensor unit consisted of visible and infrared light emitting diodes and a photodiode detector that was mounted on a plastic clip padded with foam. This sensor is illustrated in Figure 2. The interface module provided continuous analog output of the plethysmographic pulse waveform. The Criticare model 503 does not recalibrate once the initial signal is found (unless the signal is lost and the sensor repositioned) as do certain other brands of pulse oximeters. It provides a "non-gained" waveform output [25]. This quality proved to be essential in allowing detection and analysis of relative changes in the pulse waveform signal shape over time.

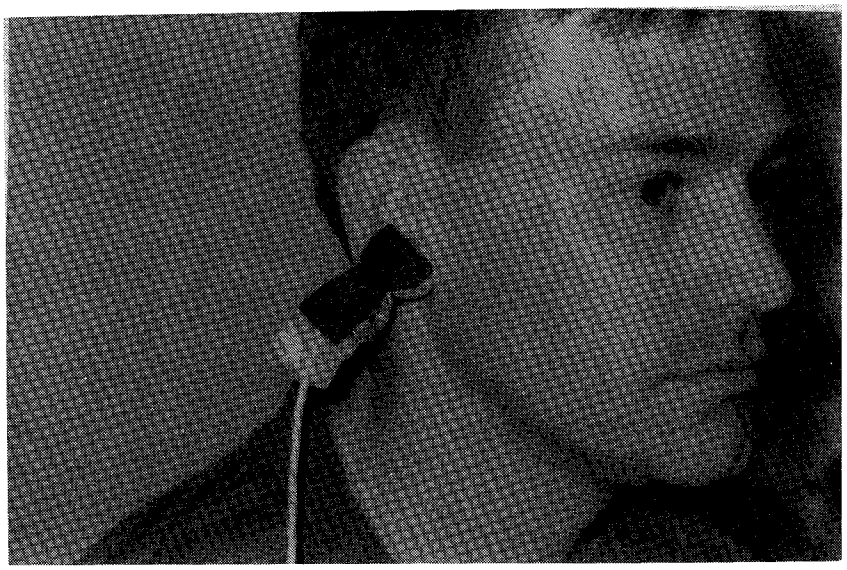


Figure 2. Ear Clip Oxysensor

A modified Sanyo 80286 12 MHz microcomputer with an 80287 math co-processor was utilized as the main platform. A Willow Peripherals video card with NTSC video capabilities provided the necessary video output, and a National Instruments multifunction I/O board (model Lab-PC) with analog-to-digital conversion capability allowed computer input of the analog signals from the pulse oximeter (pulse waveform, oxygen saturation), and analog pressure gauge, were combined and displayed onto a separate screen using a Shintron Chromatic model 370 video special-effects generator system.

A computer program was developed, written, and compiled in the

Microsoft QuickC™ language to continuously monitor, display, analyze, and record the confluence of digital and analog data being generated in real-time. The plethysmographic pulse waveform was continuously displayed and analyzed by an area-under-the-curve algorithm. In addition, pulse duration and amplitude were calculated. The pulse waveform was sampled in real-time at 100 Hz in order to determine the area, amplitude, and duration. Data samples were recorded by the computer at one-second intervals throughout the experiment and ultimately saved on a hard disk drive. After the experiment, the data were backed up onto 5.25" floppy disks.

All persons in the experimental environment (including subjects) wore standard disposable foam E-A-R™ plugs for hearing protection because the vacuum source generated more than 74 dB of noise.

Experimental Design

This investigation was conducted in two phases, requiring a total of two visits by each subject. First, a "training phase", which involved familiarization of the subject with the experiment and a trial run to determine LBNP tolerance. This also helped to reduce anxiety during the actual experimental run. During the "training phase", a standard F-4 Phantom aircraft seat (12 degree seat-back-angle from vertical) was utilized. Figure 3 illustrates the experimental setup used in this study.

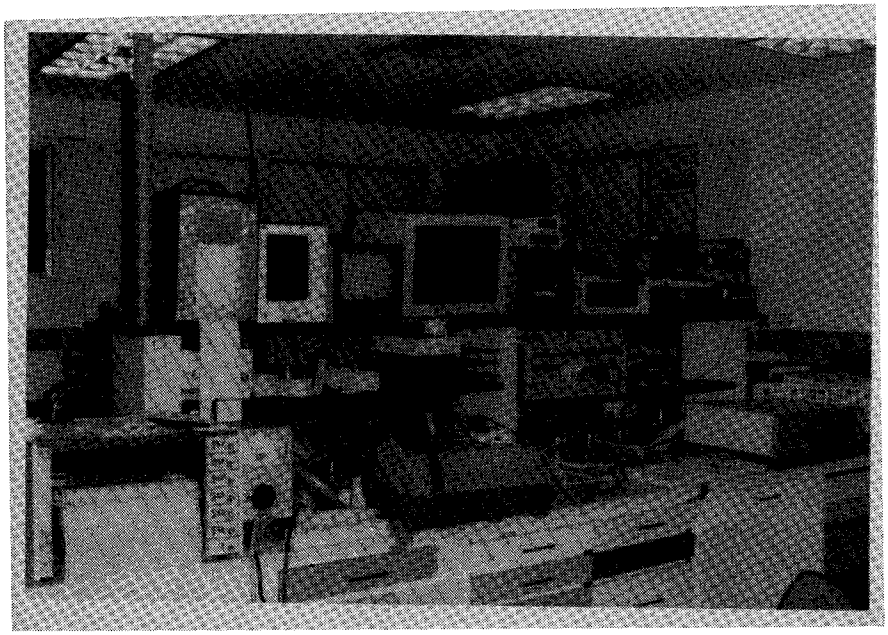


Figure 3. Experimental Setup

Since all ten subjects successfully completed the "training phase" profile and reached a minimum of -50 mm Hg of LBNP without presyncopal symptoms, all were allowed to proceed to the "experimental phase". This involved exposure to LBNP while in a standing position, which increased the pooling effect of LBNP due to the increased influence of gravity.

The "experimental phase" was the actual test and data collection run. It was conducted identically to the "training phase", except that the subject was in a standing position. In addition, the full negative pressure profile was followed until the subject experienced presyncopal symptoms or the maximum time interval was reached. There was a minimum of three days between the "training phase" and "experimental phase" for each subject.

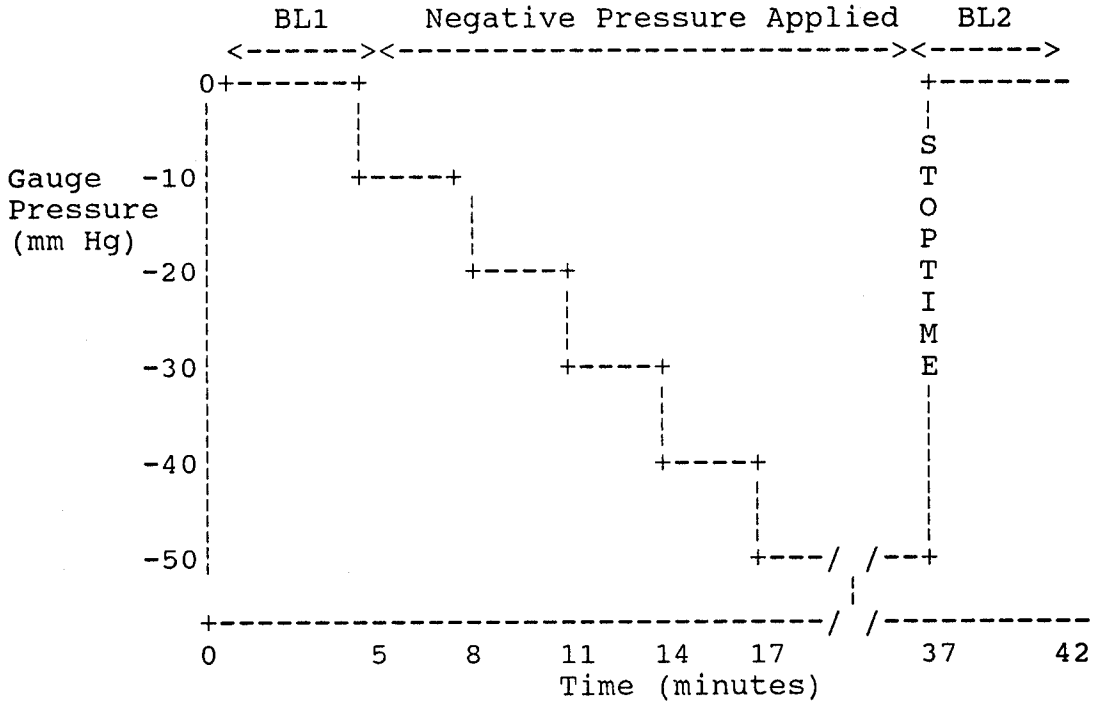
No attempt was made to control diet or fluid intake prior to the experiment. Subjects were asked to maintain their regular dietary and exercise regimens prior to the study. To protect their skin from abrasions, each subject wore either a flight suit or long underwear inside of the LBNP suit.

Foam padding was placed around the ankles to provide protection from the inside hard shell of the suit rubbing the subject's ankles. The padding also prevented damage to the outer covering material while at negative pressure by preventing the suit material from being pulled up into the leg sections. The subject donned the appropriately sized suit while either wearing shoes or molded fiberglass casting material "foot protectors." These fiberglass boots were heavily padded on the sole with foam and a sorbothane™ insert. A three-dimensional semi-rigid nylon material was wrapped around the waist area and inserted into each leg section from the top down, providing a means to equalize the pressure between leg sections. Foam padding was inserted into the leg sections after being wrapped around the waist area and thigh to provide protection for the subject and the suit material. Circular neoprene rubber tubes were placed around the subject's waist inside of the outer covering to hold the three-dimensional nylon material in place and to provide a small amount of pressure distribution. The waist seal was accomplished by placing the neoprene-impregnated nylon material (which was the external layer of the entire suit) directly against the skin, followed by an elastic bandage wrap. The seal was located just above the level of the iliac crests bilaterally. The appropriate evacuation hose and pressure port tubing was attached and secured in place.

ECG electrodes were placed in a standard Einthoven Lead II configuration, and a rhythm strip was obtained. Foam ear plugs were placed in both ears. The optimal positions for the ear clip (pulse oximeter sensor) were located. Prior to placement of the ear clip, the ear was cleansed with an alcohol pad.

After all instruments were in position, the subject was asked to assume a standing position. All instruments were then monitored and recalibrated or repositioned if necessary. Appropriate adjustments were made to the computer program parameters to allow appropriate tracking of the pulse waveform. This consisted of setting the "maximum beats per minute" parameter to within 10-20 beats of the subject's baseline heart rate, allowing proper synchronization of the computer program with the subject's pulse waveform.

After appropriate examination and authorization by the medical monitor, the experiment commenced. This consisted of the following pressure profile:



Abbreviations Used:
BL1 = Baseline 1
BL2 = Baseline 2
STOPTIME = Experimental Endpoint (maximum 37 minutes)
mm Hg = millimeters of mercury

Figure 4. LBNP Experimental Profile

The first five minutes at ambient pressure (zero mm Hg gauge pressure) were considered "baseline" data. The LBNP profile continued until the subject experienced presyncopal symptoms, requested to stop for any reason, medical profile termination criteria were met, the maximum time interval was reached, or by

request of the experimenters or medical monitor.

Each subject was observed by a physician throughout the experiment.

The following medical LBNP termination criteria, slightly modified from those originally developed at the Johnson Space Center in Houston, Texas [20], were utilized during this research:

1) Sudden decrease in systolic blood pressure > 25 mm Hg per minute, or decrease in diastolic blood pressure > 15 mm Hg per minute.

2) Sudden decrease in heart rate > 15 beats per minute.

3) Subject nausea, clammy skin, profuse sweating, or pallor of the skin.

4) Subject request for any reason.

5) Systolic blood pressure < 70 mm Hg.

6) Any significant cardiac arrhythmias/dysrhythmias, including bradyarrhythmias, tachyarrhythmias, or heart block.

7) Premature Ventricular Complexes (PVC's) meeting any of the following criteria: 6 or more PVC's per minute, R on T phenomenon, closely coupled PVC's, couplets, runs, or multifocal PVC's.

8) Heart rate (H.R.) greater than 90% of the estimated maximum as determined by the formula: Maximum H.R. = (220 - Age).

9) Loss of ECG signal for any reason.

Figure 5. Medical Criteria for Terminating LBNP Test

The medical criteria were interpreted by the medical monitor(s) on a real-time basis, and consideration was given to the current condition of the subject, trends of the measured physiological variables, and the limitations and idiosyncracies of the monitoring equipment (such as motion artifact, recalibration, sensor drift, etc.).

After the negative pressure was discontinued, an additional five minutes of post-LBNP baseline data were collected. If the subject was presyncopal, he/she was placed in a seated position. If the subject endured the entire profile, he/she remained in a standing position for these five minutes. After the five minutes

of additional data were collected, the instruments were turned off and removed in the reverse manner of how they were placed. During the entire experiment, any subjective symptoms were recorded and the time of occurrence noted.

Presyncopal subjects were questioned about the nature of their symptoms and the reason for aborting the run. After removal of the suit, the subject was evaluated by the medical monitor and allowed to leave.

Data Collection and Analysis

Data were recorded on microcomputer disk. In addition, hard copy strip charts were obtained as needed for ECG, waveform, suit pressure, and pulse waveform. The microcomputer recorded data on a hard disk drive in the following format at one-second intervals: (sample data are shown in Figure 6).

Subject:_____ Run: _____
Comment:

Ev	Time	Suit	Area	Ampl.	Dur.
0	00:00	0.30	5.10	0.13	550
0	00:01	-5.60	5.08	0.11	560
1	00:02	-8.70	5.09	0.10	560
1	00:03	-9.89	5.05	0.11	565

Figure 6. Computer Data File Format

The following describes the meaning of each column and abbreviation used in Figure 6.

Description of Columns in Figure 6:
Ev = optional Event marker (0, 1, 2, etc.)
Time = time since start of experiment in minutes:seconds
Suit = suit gauge pressure in millimeters of mercury
Area = area under the pulsatile signal, defined as the sum of all voltage readings made during a measured pulse (sampled at 100 Hertz), with the voltage waveform "area" under the pulse's minimum value removed. Since pulse signals were not synchronized with the 1 second data recording interval, this represented the latest completed pulse. This area will vary among subjects, as it depends on physical positioning of the sensor. Pulse waveforms of differing area can be obtained by simply placing the sensor in a new physical location. It is, however, stable for any given subject assuming no change in sensor position or motion artifact. It is not comparable between subjects except as a percentage change.
Ampl = the measured peak voltage value minus the measured minimum voltage of the latest completed pulse. Like the pulse area, it

is dependent upon sensor position and not comparable between subjects except as a percentage change. It is, however, stable for any given subject.
Dur = duration, in milliseconds, of the latest perceived pulse. This value is inversely related to the subject's heart rate which is also described in Figure 7.

The pulse oximeter data presented additional challenges in analysis. The pulse oximeter sensor was extremely sensitive to motion artifact, due to its physical location and nature of measurement. Despite all efforts, it was impossible to completely eliminate motion artifact from the data (due to subject head motion, verbalization, etc.). In addition, the computer program was designed to be extremely sensitive and to sample the data at 100 Hz in order to provide the highest possible accuracy in calculating the area under the pulse waveform. Unfortunately, this also created a problem in that minor fluctuations of voltage levels were occasionally incorrectly interpreted as the start of a new pulse. This comprised only a very small amount of the actual data collected.

If the pulse was not being correctly tracked, it was clearly evident on the real-time computer display and documented on the videotaped copy of the entire experiment. If the tracking problem was severe, the operator could manually adjust a synchronization parameter to again regain proper waveform tracking. The analog pulse waveform signal was continuously displayed and monitored throughout the experiment, and the computer program placed vertical lines where it currently defined the beginning and end of a pulse, as outlined below:

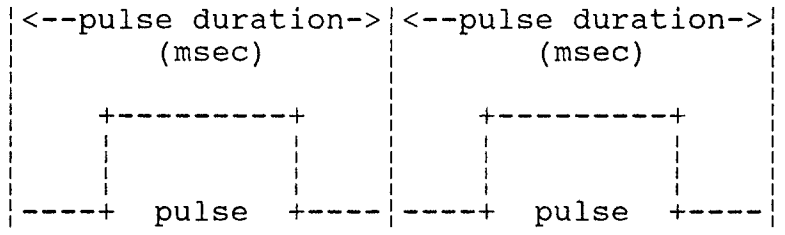


Figure 7. Description of Pulse Waveform

The pulse waveform data were susceptible to motion artifact and occasional incorrect sensing during the experiment. Fortunately, this occurrence only comprised a small amount of the total data but did cause several areas of artifactual figures.

Examples of Generating Incorrect Pulse Waveform Data

Motion artifact occasionally caused the computer program to miss

the actual endpoint of the current pulse, creating an artificially large "perceived pulse" having more than twice the duration and area of the most recent pulse. Motion artifact occasionally caused "spikes" in the pulse waveform voltages, causing the computer to interpret that the end of a pulse had been reached and therefore creating an artificially small "perceived" pulse of extremely small duration and/or area (temporarily).

Temporary loss of the sensor signal due to motion artifact occasionally caused the sudden onset of an extremely small pulse amplitude and/or impossibly low oxygen saturation (for example 50%). This resolved upon discontinuation of motion, again allowing proper synchronization with the pulse signal.

RESULTS

Results of LBNP Profile

All ten subjects completed the seated "training phase" uneventfully and without presyncopal symptoms. This involved, at minimum, reaching the maximum level of -50 mm Hg of LBNP. During the experimental (data collection) phase, subject 05 became presyncopal before the onset of any pressure and before any data were recorded. This subject repeated the experiment approximately one week later and had presyncopal symptoms after application of the normal LBNP pressure profile. Of the ten subjects who participated in the experimental phase (standing LBNP), six developed presyncopal symptoms. Four of the ten subjects endured the entire 42 minute profile without presyncopal symptoms. In the group of six presyncopal subjects, three were male and three were female. The most common non-presyncopal complaints were foot discomfort, fatigue from standing, and boredom. All experimental runs that were aborted due to presyncopal symptoms were stopped by subject request. The most common presyncopal symptoms included: stomach awareness, mild nausea, lightheadedness, sensation of impending syncope, hot/cold flashes, feeling cold & "clammy," and sweating.

A difference in LBNP tolerance was noticed between males and females in this experiment. Since there were very few subjects and hydration status was not controlled, no attempt was made to analyze this difference. All three female subjects became presyncopal. Two of the three females became presyncopal at -40 mm Hg, before reaching the maximum negative pressure level. Only three of the seven males experienced presyncopal symptoms. All three of the presyncopal males reached the maximum level of -50 mm Hg before experiencing presyncopal symptoms. Previous work by Frey et al., demonstrated no qualitative differences in cardio-vascular responses to LBNP between men and women [2, 3], so these observations were not felt to be significant.

During the experimental run of subject 06, a technical problem occurred with the pulse oximeter sensor, causing erroneous data just prior to the experimental endpoint. It is believed that a positional change in the physical location of the pulse oximeter sensor occurred, which caused a sudden, extremely large, and grossly abnormal increase in pulse waveform area and amplitude prior to discontinuation of pressure. This abnormality is clearly documented on the videotape and computer data file, and did not occur in any of the other subjects. This sudden increase was inconsistent with physiological expectations. Consequently, the following statistical analysis excludes the pulse oximeter data obtained for subject 06. With respect to symptoms they experienced, three of the six presyncopal subjects (subjects 02, 05, and 10) spontaneously reported similarity to +Gz exposure after completing their experimental run. The following table summarizes the results of the LBNP profile:

Table 2. List of Subjects and Results of LBNP Profiles

SUBJECT ID	PRE-AGE	STOP-SYNCOPE?	TIME	SYMPTOMS EXPERIENCED BY SUBJECT
01	38	YES	27:28	mild nausea, cold, clammy, short of breath, lightheaded, feeling of impending syncope
02	31	YES	20:16	stomach awareness, nausea, sweaty, faint/lightheaded
03	31	NO	37:02	foot pain, brief nausea (mild)
04	30	NO	37:00	pressure on feet, legs feeling "heavy"
05	25	YES	15:27	stomach awareness, hot flash, very faint, near-syncope, visual dimming
06	25	YES	15:17	mild nausea, cold, sweating, lightheaded, near-syncope
07	30	NO	37:02	brief stomach awareness, sweating
08	41	NO	37:00	foot discomfort, boredom
09	29	YES	36:01	mild nausea, sweaty, "clammy" lightheaded, short of breath, fatigue
10	26	YES	24:58	disoriented, fatigue, anxiety, high workload, lightheaded, sweaty, felt impending syncope and asked to stop

Note:
STOPTIME is in minutes:seconds and represents the point at which the LBNP profile was discontinued.

This was due to subject request, medical termination criteria, or achieving the full profile without presyncopal symptoms.

Statistical Results

For all subjects, the physiological variables were compared in the following manner: (Ref. Figure 4)

BASELINE 1 VALUE (BL1) = The median (pulse area, amplitude, duration), value of the last 15 seconds of BL1.

STOP VALUE = The median (pulse area, amplitude, duration), value of the 15 seconds prior to STOPTIME.

BASELINE 2 VALUE (BL2) = The median (pulse area, amplitude, duration), value of the last 15 seconds of POST-BASELINE.

All three physiological variables were analyzed as a percent change from BL1. For ease of interpretation, the plots which follow are scaled as a percent of Baseline 1 (i.e., BL1 represents 100%). Using the percentage change from BL1 (as opposed to the actual value) eliminated erroneous comparisons of values. Otherwise, a pulse area change from 9 to 7 would have been similarly interpreted as a change from 3 to 1 (i.e., difference of 2 in both cases but a large difference in percent change between them).

Because the pulse oximeter data had occasional motion artifact which would cause extreme values, the median of each 15 second sampling period for pulse area, amplitude, and duration was used instead of the mean. As a measure of central tendency, the median is less sensitive to extreme values than the mean.

The Student's t-test was used to determine the significance of percent change from BL1. Each of the resulting t-tests used $p = 0.05$. In addition, a two-sample t-test with $p = 0.05$ was used to compare the percent change from BL1 between the presyncopal and non-presyncopal groups at STOPTIME.

Because of the nature of this approach and the relatively small sample sizes (N = 4, 5, or 6), the results were cautiously interpreted. Table 3 shows the p-values calculated using percent change from (BL1) for each physiological variable.

Table 3. Comparisons Within Groups (P-VALUES)

		NON-PRESYNCOPAL GROUP		PRESYNCOPAL GROUP	
Variable	Comparison	P-Value	Mean Δ%	P-Value	Mean Δ%
AREA	BL1 to STOP	.0208	-56.86%	.0048	-44.00%
	BL1 to BL2	.0100	-38.33%	.6997	+13.60%
AMPLITUDE	BL1 to STOP	.0280	-53.75%	.0236	-38.27%
	BL1 to BL2	.0070	-46.12%	.5716	-13.49%
DURATION	BL1 to STOP	.0030	-19.62%	.3145	-11.48%
	BL1 to BL2	.8768	-0.91%	.0020	+23.72%

Notes:
BL1 = Pre-Baseline or Baseline 1
STOP = STOPTIME (experimental endpoint)
BL2 = Post-Baseline or Baseline 2
N=4 for the non-presyncopal group
N=5 for the presyncopal group: AREA, AMPLITUDE, DURATION,
Mean Δ% = mean change in percent for indicated comparison.

Example: For AREA, the mean for the non-presyncopal group at STOPTIME was 56.86% less than the value at BL1.

Table 4 shows the p-values obtained from a two-sample t-test by comparing the percent change from BL1 at STOPTIME between the presyncopal and non-presyncopal groups.

Table 4. Comparisons Between Groups at STOPTIME

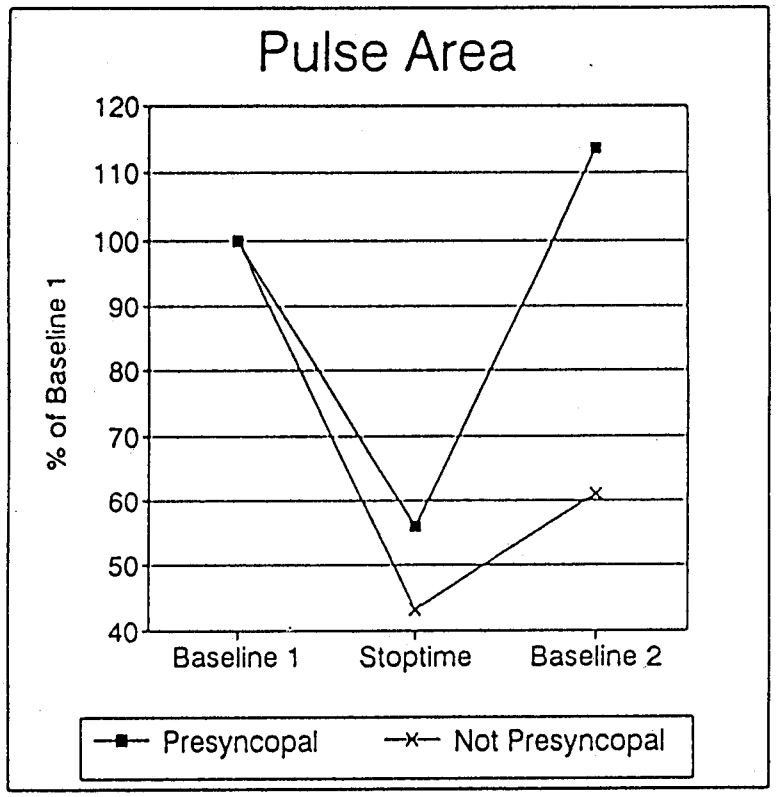
Variable	P-Value	Mean Δ%	Pooled S.D.
AREA	.3957	+12.87%	21.20%
AMPLITUDE	.3920	+15.48%	25.30%
DURATION	.5015	+8.14%	17.13%

Notes:
N=4 for the non-presyncopal group
N=5 for the presyncopal group: AREA, AMPLITUDE, DURATION
S.D. = Standard Deviation
Mean Δ% = mean change in percent, between groups, of the variable at STOPTIME

Example: At STOPTIME, for the variable AREA, the group mean for the presyncopal group was 12.87% greater than the group mean for the non-presyncopal group.

Figures 8 through 10 illustrate the results for each of the three variables (pulse area, pulse amplitude, pulse duration). All values are displayed as a percent of BL1.

Each figure contains two graphs. The upper line graph shows the group mean at each experimental sampling point (BL1, STOPTIME, and BL2). The lower graph shows the same values, with the whiskers extending from each bar representing the 95% confidence interval for the means.



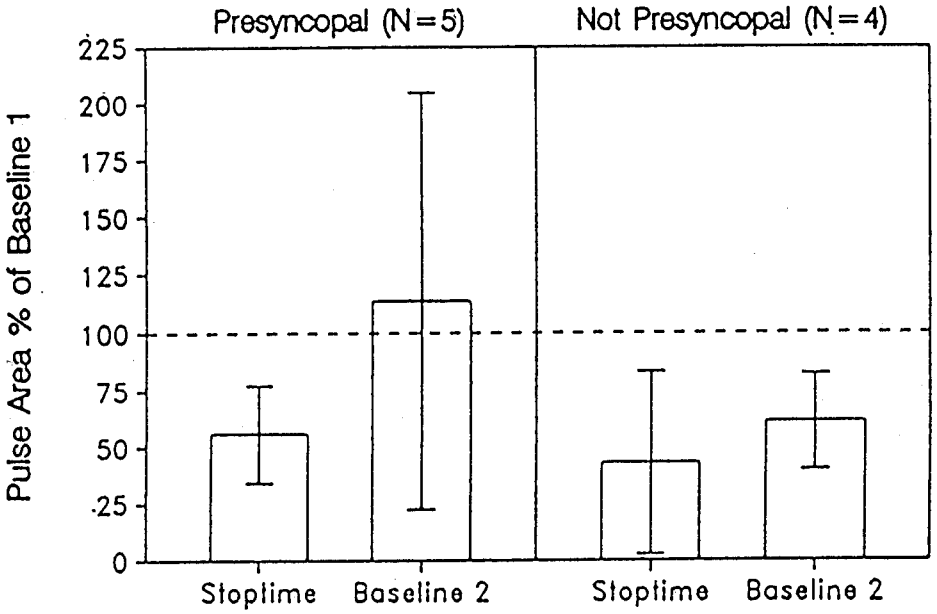
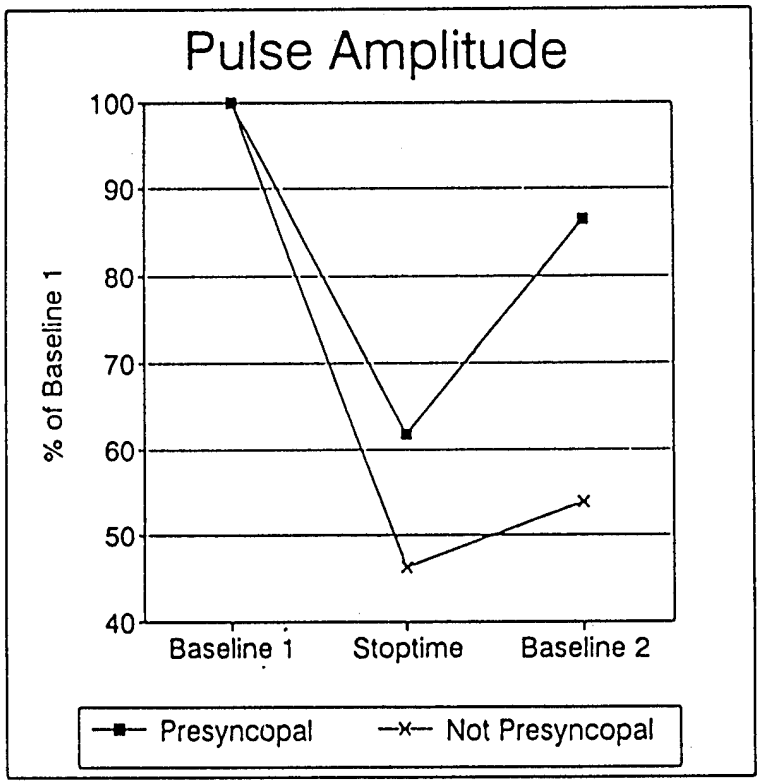


Figure 8. Pulse Area



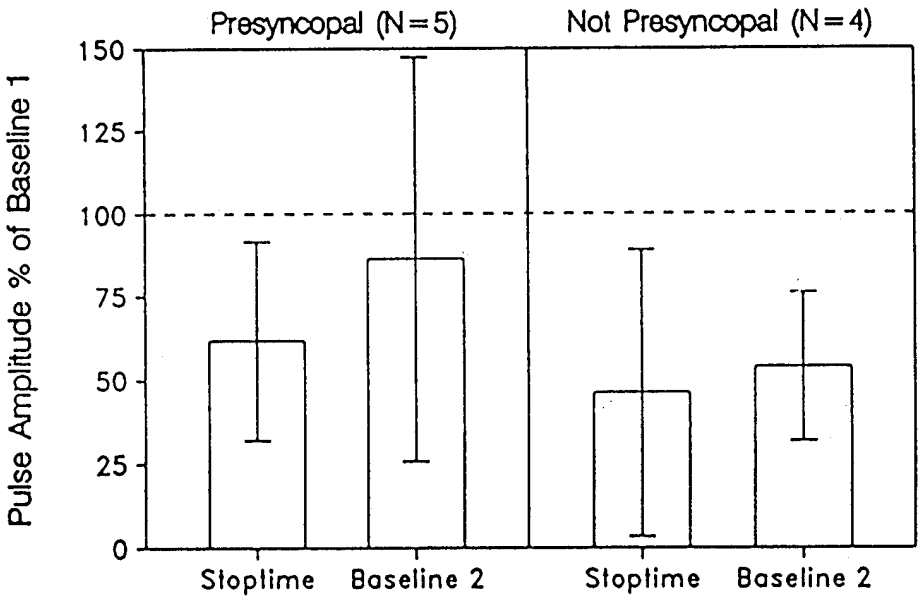
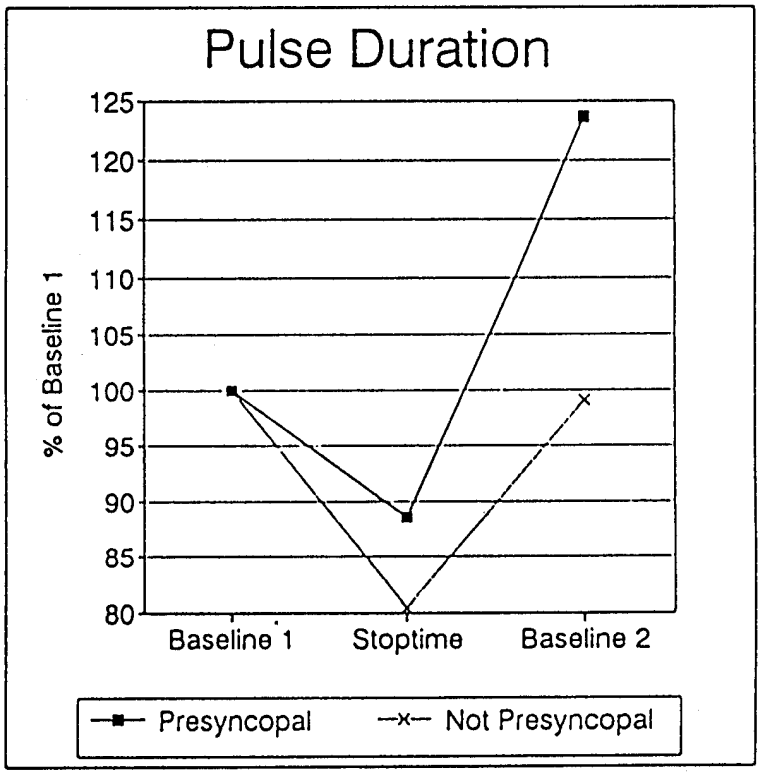


Figure 9. Pulse Amplitude



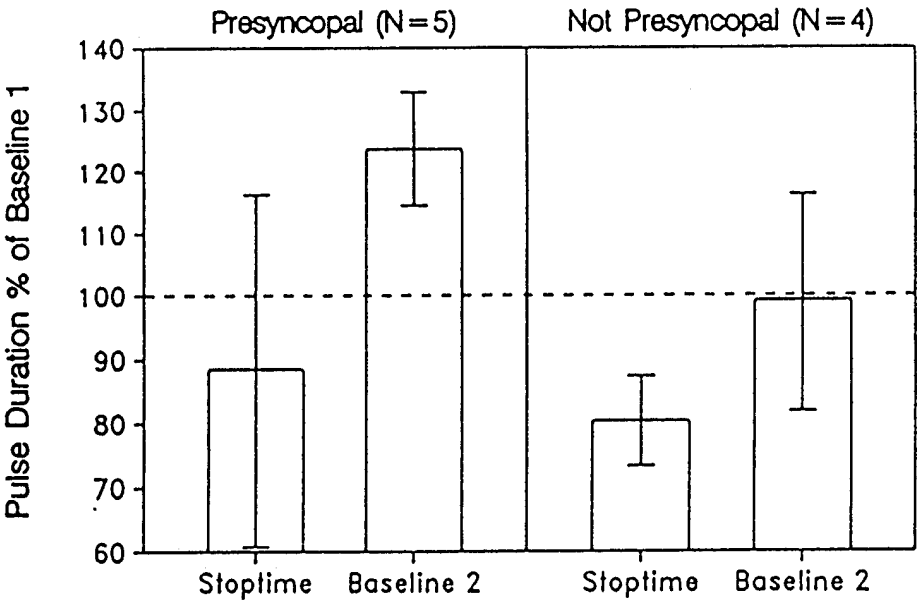


Figure 10. Pulse Duration Graphs: Percent of Baseline 1

The following plots in Figure 11 show each variable, displayed as a percent of BL1, at STOPTIME (experimental endpoint) and BL2. Both groups are simultaneously displayed. BL1 represents 100%. In addition, the y-axis scale is identical in all graphs.

N = 4 for all variables involving the non-presyncopal group.
N = 5 for the presyncopal group variables AREA, AMPLITUDE, DURATION.

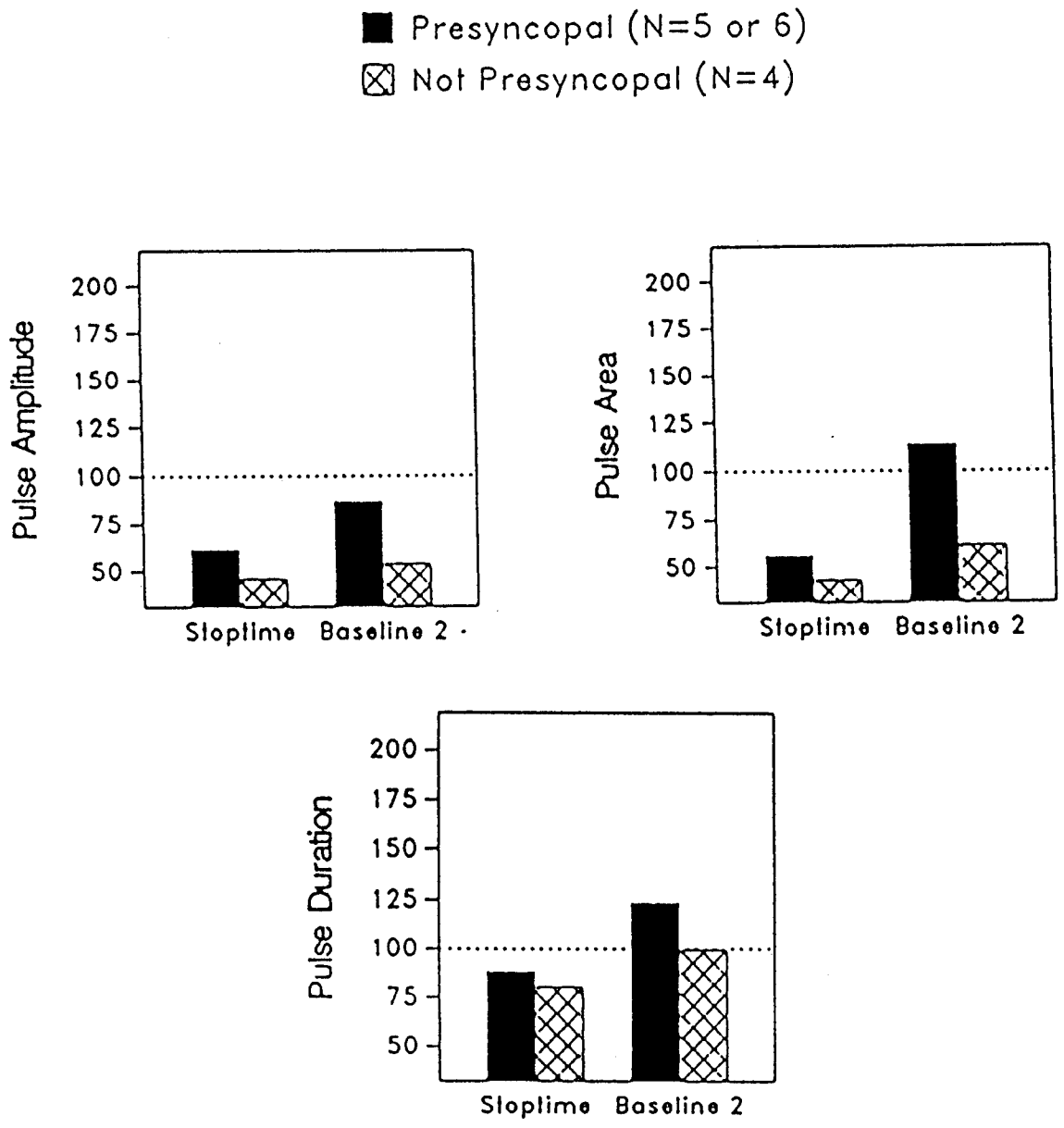


Figure 11. Mean Percent of Baseline 1

DISCUSSION

Pulse Waveform During LBNP

Changes in the pulse oximeter waveform signal have several potential causes. Ultimately, they include any changes which modify the amount of infrared and red light absorbed as detected by the sensor [22, 23, 24, 25]. Since the amount of tissue and bone remains constant, the amount of light absorbed by blood is the variable. Potential factors which could alter this analog signal waveform include sensor clip positional change, ear tissue compression by the sensor clip, changes peripheral vasoconstriction, intravascular blood volume changes, and motion artifact. Sensor clip positional change was not felt to play a significant role because the subjects remained in a stationary position. Multiple repeated test measurements during the design of this experiment for significant periods of time at ambient pressure did not demonstrate the same changes that occurred during LBNP, making tissue compression an unlikely influence. In addition, the fact that the pulse waveform signal amplitude consistently increased after discontinuation of LBNP indicated the change was because of blood flow, not artifact. Since the sensor measures spectrophotometric absorption, a change in the oxygen saturation of the blood could also change the shape of the pulsatile signal.

Pulse Waveform Area During LBNP

The analog pulse waveform area exhibited relative stability during the baseline portion of the LBNP profile. Upon exposure of the subject to negative pressure, the pulse waveform area began to gradually decrease, consistent with pooling of blood in the lower one-half of the body and a decreased central blood volume. After discontinuation of negative pressure, it increased toward the original steady state baseline. In several subjects, it exhibited an "overshoot," consistent with a "reactive hyperemic" response of the peripheral or cerebral circulation.

The pulse waveform area decreased below the initial baseline in both the presyncopal and non-presyncopal groups. Overall, the percentage decrease was greater in the non-presyncopal group (56.86% mean percent decrease vs. 43.99% in the presyncopal group). However, this small difference must be cautiously interpreted because of the small number of subjects in each group. It may also reflect differences in LBNP tolerance.

It is important to realize that both of the groups were exposed to the same negative pressure profile and therefore experienced the physiological effects of LBNP. This explains why pulse waveform area decreased significantly in both groups. The decrease in pulse area during LBNP is consistent with the decrease in pulse area during LBNP is consistent with the

physiological pooling of blood in the lower one-half of the body, away from the upper body and head where the sensor was located. The pulse waveform area decrease was not a smooth gradual curve. Motion artifact was easily identifiable by sudden, extremely large changes in pulse area which ceased upon discontinuation of movement. In addition, while the overall trend was a continual decrease, small increases followed by decreases were also continuously apparent in the data. These data are illustrated in Figures 12 and 13.

The observed effect may be partly related to a phenomenon observed by Buick et al., using ear opacity techniques during exposure to gradual onset +Gz [19]. They observed cyclical changes in the ear opacity pulse with a mean cycling period of 10.4 seconds [19]. The mean difference in opacity level within cycles was 17.1% of the +1 Gz opacity value [19]. Their results suggested that head-level perfusion may not be constant during sustained +Gz [19].

Pulse Waveform Amplitude During LBNP

The pulse waveform amplitude demonstrated changes similar to pulse waveform area. After a stable baseline, the amplitude began to gradually decrease upon application of LBNP. Upon discontinuation of the pressure, this amplitude again returned toward the original baseline value.

The pulse waveform amplitude likewise demonstrated a significant decrease relative to the initial baseline in both the presyncopal and non-presyncopal groups. Overall, the percentage decrease was greater in the non-presyncopal group (53.75 mean percent decrease vs. 38.27% decrease in the presyncopal group). This might be explained by a difference in tolerance to LBNP between the groups. It is important to remember that both groups were exposed to LBNP and therefore experienced the pooling of blood associated with LBNP. The decrease in pulse amplitude during LBNP is consistent with the physiological pooling of blood in the lower one-half of the body, away from the upper body and head where the sensor was located.

Pulse Duration During LBNP

As previously discussed and described in figure 7, the pulse duration represents the time interval (measured in milliseconds) from the beginning to the end of a particular pulse waveform.

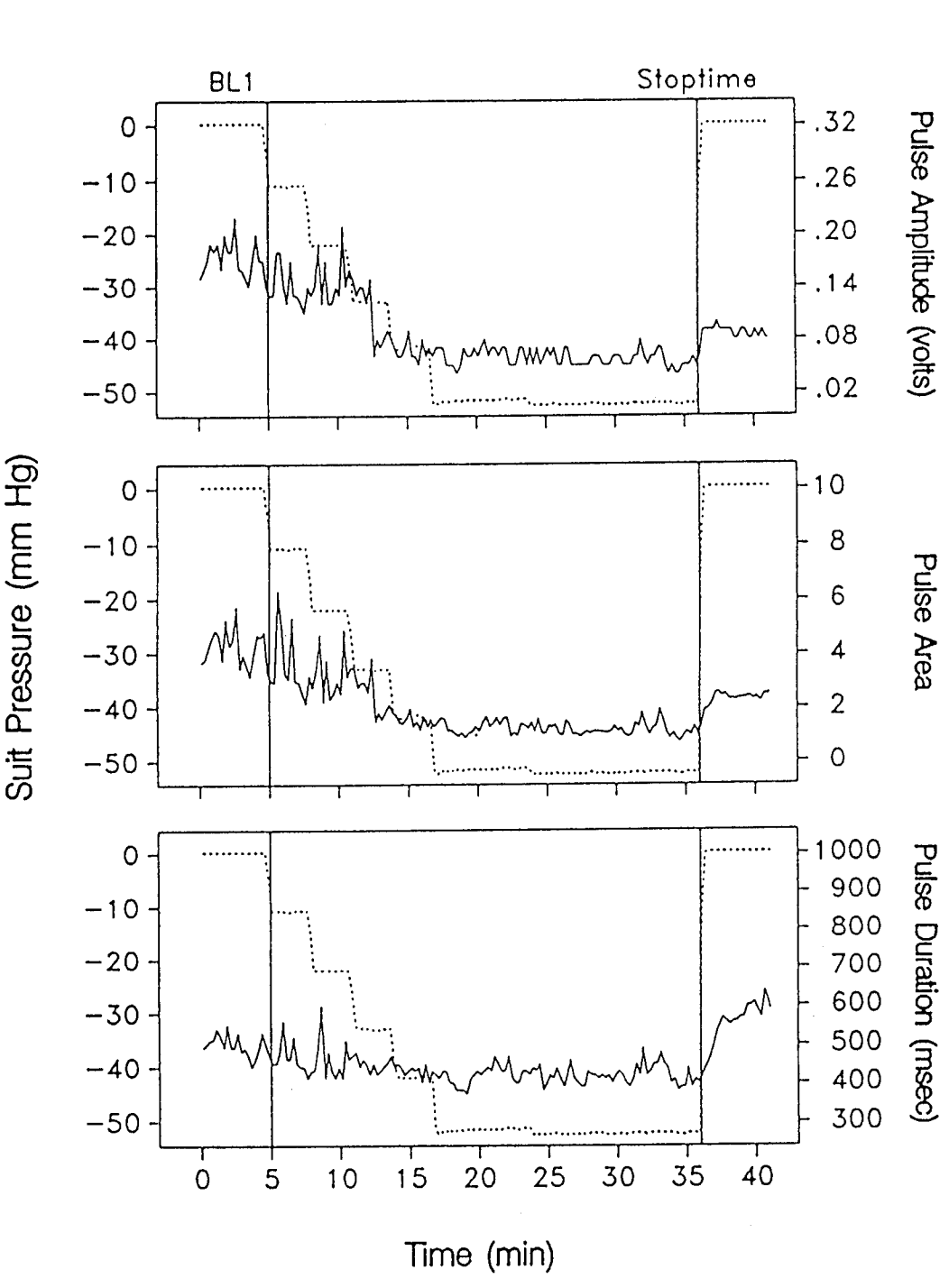


Figure 12. Presyncopal Pulse Amplitude, Pulse Area, and Duration (Subject 09)

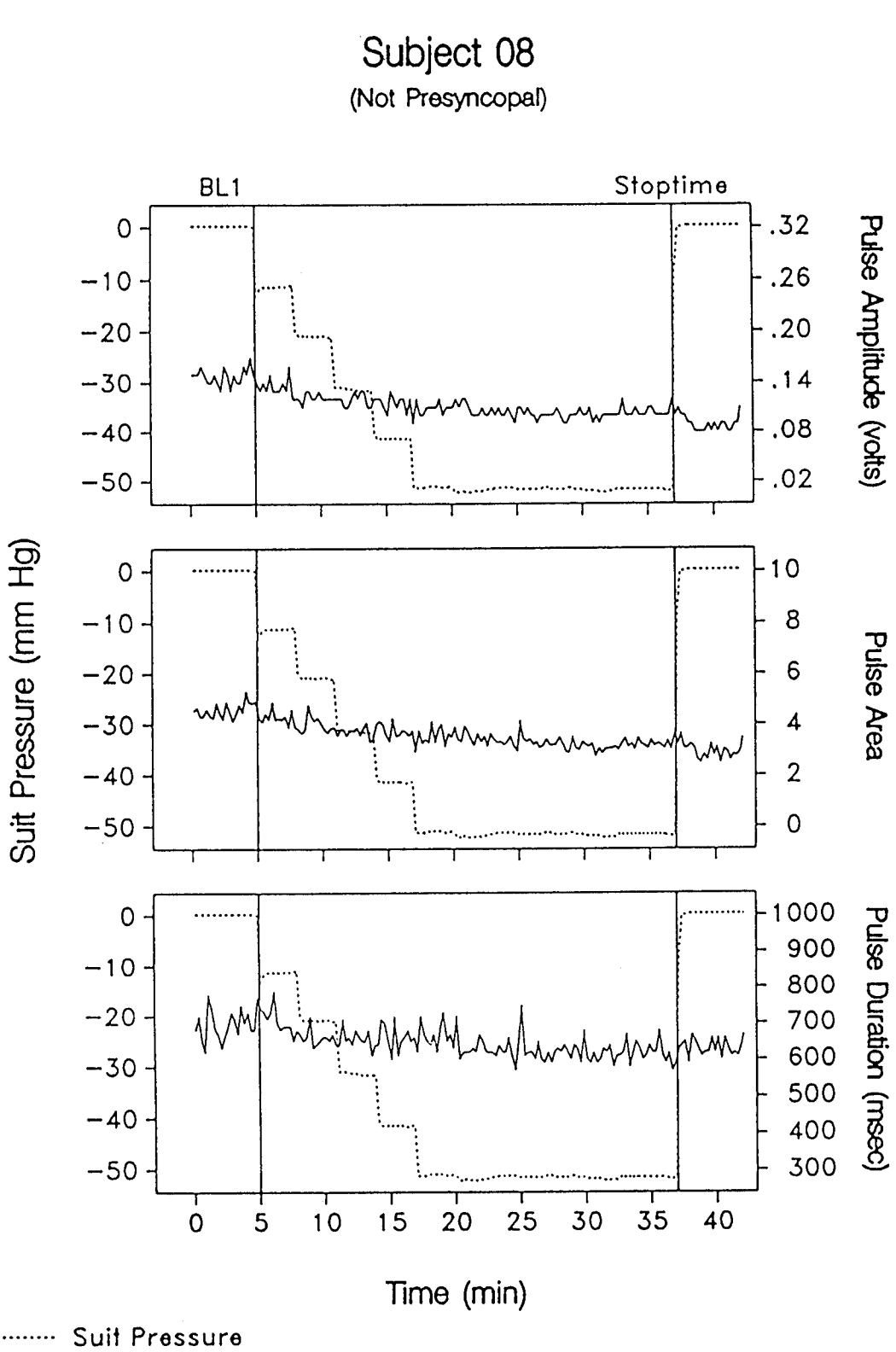


Figure 13. Non-Presyncopal Pulse Amplitude Pulse Area, and Pulse Duration (Subject 08)

Pulse duration is inversely related to heart rate. Faster heart rates result in a greater number of pulse waveforms per unit time, therefore the pulse duration is smaller. Slower heart rates will have a longer pulse duration value.

Overall, both groups demonstrated a decrease in pulse duration at the experimental endpoint, consistent with an increase in heart rate. The actual percent change from baseline at one endpoint was only significant in the non-presyncopal group. Upon examination of the values for each individual subject, all non-presyncopal subjects and 4 of the 5 presyncopal subjects (for whom duration values were analyzed) demonstrated a decrease in pulse duration. This is consistent with an increase in heart rate prior to the endpoint. Because of the small group size (N = 5) in the presyncopal group, the effect of one subject's (subject 05) increase in pulse duration (decrease in heart rate) at the endpoint effectively cancelled out the statistical significance of the other 4 subjects in that group.

The increase in heart rate in all but one subject is consistent physiologically with the increased workload experienced by all subjects during this exposure to LBNP. In addition, it may also be partly explained by a tachycardic response to decreased central blood volume. The one subject (subject 05) who actually demonstrated a decrease in heart rate at the endpoint may have been experiencing a bradycardic or vasovagal response with impending cardiovascular decompensation. This subject, in fact, did experience the most significant presyncopal symptoms of the entire group.

Return To Baseline Values

The six variables previously discussed were also analyzed as to whether or not they returned toward the Baseline 1 value after discontinuation of LBNP. This was performed by comparing the percent change from BL1 to BL2 using a Student's t-test, with an alpha value of 0.05.

In the non-presyncopal group, Pulse Duration demonstrated no significant difference between BL1 and BL2 indicating a return to baseline.

Also in the non-presyncopal group, Pulse Area and Pulse Amplitude all demonstrated a trend toward the original baseline, but remained statistically different, indicating a lack of return to baseline. It should be emphasized that a definite trend was apparent, and that these variables were measured only five minutes after discontinuation of LBNP. It is presumed that had these variables been measured again in five or ten minutes, they would have shown a continuing trend and perhaps a complete return to their original baseline values. In addition, the non-

presyncopal subjects remained standing during the five minutes of BL2, whereas the presyncopal subjects assumed a seated position. This most likely influenced blood and interstitial fluid return, slowing the recovery to baseline after LBNP.

In the presyncopal group, all variables (amplitude and area) except for pulse duration demonstrated a return to baseline. Pulse duration was longer at BL2 than at BL1 in the presyncopal group, indicating that heart rate was slower. This was statistically significant ($p=0.002$). Duration is inversely related to heart rate. A faster heart rate will cause a larger number of pulses per unit time, and consequently a smaller time interval per pulse. At the experimental endpoint, pulse duration actually decreased below its BL1 value indicating an increase in heart rate.

After discontinuation of pressure, the pulse duration again increased toward the BL1 value and actually surpassed the original value. In other words, the group mean heart rate was lower at BL2 than at BL1.

Differences Between Groups at Stoptime

As previously discussed, a two-sample t-test was performed to detect a difference between the presyncopal and non-presyncopal groups at the experimental endpoint (STOPTIME). The resulting P-values were previously listed in Table 4. The variables' pulse area, pulse amplitude, and pulse duration did not demonstrate a statistically significant difference between groups at the endpoint. It must again be emphasized that both groups experienced the physiological effects and pooling of blood caused by LBNP.

Warning Interval

Changes in several of the variables measured provided a "warning interval" just prior to subject abort/presyncope. The "warning interval" was defined as "the onset of a significant trend in a physiological variable, occurring prior to the presyncopal endpoint, which continued until the endpoint was reached, and reversed itself upon discontinuation of LBNP."

The warning interval for the pulse waveform tended to gradually decrease in area and amplitude upon application of negative pressure, with a minimal value at the point of presyncope. The pulse waveform subsequently returned toward baseline after discontinuation of the negative pressure. This effect is presumably related to the gradual pooling of blood in the lower one-half of the body. Peripheral vasoconstriction may have also played a role in this phenomenon. Since the pulse waveform

sensor site was the ear lobe, it did not directly measure deep cerebral blood flow.

All subjects above experienced presyncopal symptoms and were unaware of any changes seen on the monitoring devices. Trends for pulse waveform area and amplitude began with the onset of LBNP, and reversed upon discontinuation of negative pressure.

Subjective Nature of Presyncopal Symptoms

Since the stop point was under subject control, the endpoints all varied depending upon the individual symptom tolerance of each subject. In other words, some subjects may have aborted sooner than others in relation to severity of symptoms.

Comments and Limitations

Because of unavoidable limitations, only ten subjects were able to be included in this study. Furthermore, only six of the ten actually reached the desired endpoint. Obviously, a larger number of subjects would be desirable for any type of statistical analysis and conclusion. The small number of subjects in each group had great impact on the ability to demonstrate statistical significance in several variables. This study was performed in a 1 G static environment. In order to validate this method of real-time analysis, a similar study should be performed analyzing the pulse waveform during exposure to +Gz.

In this experiment, all of the subjects did not reach the presyncopal endpoint. This was due to limitations of the amount of negative pressure available from the vacuum source. Ideally, a continuous pressure ramp until onset of symptoms could have been used which would have increased the number of presyncopal subjects.

In most LBNP research, the subject is in a supine or seated position. This experiment required the additional gravity gradient available in the standing position because of limitations in the vacuum source.

The site chosen for the pulse oximeter sensor was the ear lobe. The arterial blood supply to the earlobe is derived from the posterior auricular artery (a branch of the external carotid), the anterior auricular artery (a branch of the superficial temporal artery), and an arterial branch from the occipital artery [5]. Ultimately, these all arise from the external carotid artery [5]. Several other locations were evaluated, including the use of reflectance transducers which can be placed virtually anywhere on an exposed skin surface. The nasal septum, whose blood supply is derived from the internal carotid artery

through branches of the enthmoid artery [5], was considered as a possible site, but we were unable to obtain a reliable sensor configuration at this location.

In several cases, the return to baseline would have been more complete if several of the variables had been measured longer than five minutes after the experimental endpoint.

In the non-presyncopal group, the return of several variables to baseline would have been improved by having the subject assume a seated position. The presyncopal group was placed in a seated position after reaching the endpoint. This difference most likely explains the delayed return to baseline of pulse area and amplitude in the non-presyncopal group.

As previously mentioned, the pulse oximeter sensor used was extremely sensitive to motion artifact. Stabilization of the head and sensor unit would have decreased this effect. Pulse oximeters are available which utilize the R-wave of the ECG to automatically synchronize the identification of the beginning of a new pulse [25]. A similar method could be used to improve the tracking and analysis of pulse waveforms and eliminate much artifactual data.

Blood pressure and heart rate were continuously monitored throughout the experiment. These were mainly for medical monitoring purposes, and were not included in the final analysis as the responses of heart rate and blood pressure to LBNP are already well-studied. Had analysis of heart rate and blood pressure been attempted, it would have been complicated by large gaps of missing data which occurred during re-calibration of the blood pressure unit that was utilized.

System for Detecting Presyncope/GLOC

Based on the results of this experiment, it would seem that the best arrangement for a system to detect presyncope/GLOC would be a combination of physiological sensors, without relying on a single system. For example, oxygen saturation decreased sharply in some but not all subjects. Similarly, there were varying degrees of change between subjects in cerebral blood flow velocity prior to presyncope.

Physiological variables including heart rate and pulse oximeter analog waveform, all have potential applicability in such a system. If the pulse oximeter was to be used in a warning or feedback device, a stable platform would be required for the sensor to reduce the motion artifact inherent in an operational environment. The possibility of building many of these sensors into a crewmember's helmet and/or life support equipment is technically quite feasible and has already been accomplished, for

the most part, at Wright-Patterson Air Force Base [12].

CONCLUSIONS

This study evaluated a method for measuring and quantifying changes in the pulse waveform analog signal during LBNP. The intent was to simulate exposure to +Gz, and to determine if changes in any of these variables could be used to detect or predict presyncope.

Statistically significant changes in physiological variables were detected prior to termination of pressure during presyncopal LBNP.

These changes included:

1) Decreased pulse waveform area
2) Decreased pulse waveform amplitude

The pulse waveform area and amplitude, although showing significant decreases consistent with blood pooling due to exposure to LBNP, did not display any sudden changes just prior to presyncope. Instead, pulse area and amplitude decreased in a gradual trend which began with the onset of LBNP, and reversed upon discontinuation of the negative pressure.

If minimum amplitude values for each user were pre-established prior to critical monitoring periods in the ground-based acceleration environment these values could provide several potential applications of medical monitoring. These include early detection of presyncope/GLOC during Space Shuttle operations (including landing/re-entry, Space Station Assured Crew Return Vehicle re-entry), the National Aerospace Plane, +Gz exposure, and ground based or orbital LBNP experiments. This information could be used for remote medical monitoring or as physiological feedback to a pilot, crewmember, or safety control system.

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